Nitrogen Narcosis

Another danger associated with working in a hyperbaric chamber is the possible occurrence of nitrogen narcosis: the high tensions of nitrogen in blood which occur when air is inhaled at increased pressures, mainly above 6 ATA, have a narcotic action.

At 3 ATA, our working pressure, various tests were carried out in order to decide whether surgeons and anæsthetists breathing air would be adversely affected in their performances by the increased nitrogen tension in their blood. In some workers the first dive produced mild psychological changes such as a feeling of hilarity and loquacity but, after regular work in the chamber, this effect disappears. With regard to manual dexterity, there were no special difficulties associated with surgical or anæsthetic technical procedures. A study of handwritten surgical notes and anæsthetic charts made under 3 ATA pressure revealed no differences compared with those written at normal pressure. Handwriting and typewriting in air at 3 ATA during the course of four hours' exposure showed no significant changes. We conclude that air at 3 ATA for several hours has no detectable influence on psychological or manual performance in the majority of workers.

A new field for research, rather different from Boerema's original idea, has been opened up. Much is still unsolved and the situation can be compared with that surrounding hypothermia in the early 1950s. Nevertheless operations and therapies and anæsthesia using modern techniques are practical propositions in a hyperbaric chamber and many worth-while results have been achieved. Further research is necessary in order to minimize the risks and to clarify problems, particularly those associated with ventilation, blood flow and oxygen toxicity under high oxygen pressures.

REFERENCES

Behnke A R, Johnson F S, Poppen J R & Motley E P (1935) *Amer. J. Physiol.* **110**, 565

Bendixen H H, Hedley-Whyte J & Laver M B (1963) *New Engl. J. Med.* **269**, 991

Boerema I, Kroll J A, Meyne N G, Lokin E, Kroon B & Huiskes J W (1956) *Arch. Chir. Neerl.* **8**, 193

Boerema I, Meyne N G, Brummelkamp W H, Bouma S, Mensch M H, Kamermans F, Stern Hanf M & Van Aalderen W (1959) *Arch. Chir. Neerl.* **11**, 70

Boerema I, Meyne N G & Vermeulen-Cranch D M E (1962) *Surgery* **52**, 796

Churchill-Davidson I, Sanger C, Thomlinson R H (1955) *Lancet* i, 1091

Haldane J S & Priestley J G (1935) Respiration. Oxford

Kety S S (1950) *Anesthesiology* **11**, 517

Keuskamp D H G (1961) *Arch. Chir. Neerl.* **8**, 193

Meyne N G, Keuskamp D H G, Sluijter M E & Boerema I (1962) *J. cardiovasc. Surg.* **3**, 308

Meyne N G, Vermeulen-Cranch D M E, Sluijter M E, Eloff S J P, Schripsema L, Deen L, Schoemaker G & Boerema I (1962) *J. thorac. cardiovasc. Surg.* **44**, 749

Observations on Hyperbaric Oxygenation during Anæsthesia

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The therapeutic use of hyperbaric oxygen rests on two assumptions: that significant increases in tissue oxygen tensions can be produced by inhaling oxygen at supra-atmospheric pressures and that such increases in oxygen tension can produce therapeutic benefit in ischæmic or hypoxic organs. Ideally, to test critically these basic assumptions, it would be necessary to measure tissue oxygen tension in each organ both in health and in disease. Since the interpretation of measurements made by tissue oxygen electrodes is difficult, it is reasonable to accept, for the present, measurements made on venous blood, cerebrospinal fluid or urine as giving some indication of what is happening at the cellular level.

The first link in the chain that ends in the tissues is the alveolar oxygen partial pressures achieved by breathing hyperbaric oxygen. Most large pressure chambers are pressurized with air and consequently oxygen has to be administered to the patient via some form of mask, hood or helmet. At first, in both Amsterdam and Glasgow, the BLB mask was the standard method of administering oxygen. The efficiency of this system of oxygen administration (as indicated by measurements of alveolar oxygen partial pressure) is shown in Table 1, which is based on results obtained in a study of 10 volunteers breathing oxygen at 2 atmospheres from a BLB mask at a flow rate of 8 litres/minute (McDowall, Ledingham, Jacobson & Norman 1965, in preparation). The average efficiency of the BLB mask was only 63% and there were wide variations in efficiency between individual subjects as indicated by the large standard deviation. These findings agree closely with those of Kory *et al.* (1962).

The performance of the BLB mask was therefore shown to be inadequate for the administration of oxygen in the pressure chamber. Following a suggestion by Squadron Leader J Ernsting

Table 1

Comparison of BLB mask and 'test' system of oxygen administration

	Mean alveolar oxygen partial pressure	*Efficiency*
BLB	898 ± 197 mmHg	63%
Test system	1,253 ± 108 mmHg	88%

of the RAF Institute of Aviation Medicine, a new system consisting of an airline pilot's mask and a hinge-type demand valve was studied by McDowall and his co-workers.

Their results, which were based on measurements of alveolar oxygen partial pressure in 7 volunteers breathing oxygen from this 'new' system at 2 atmospheres absolute, are also given in Table 1. It will be seen that the efficiency of this system was much higher than that of the BLB mask and that the scatter of results between individual subjects was considerably less. This mask is now being used routinely for the treatment of patients in the pressure chamber.

The next possible difficulty in raising tissue oxygen tensions by hyperbaric oxygen breathing would be the existence of a large difference between the partial pressure of oxygen in the alveoli and the partial tension of oxygen in the arterial blood. Such an alveolar-arterial oxygen tension difference or gradient, if present, could only be caused by venous admixture or 'shunting' at the high levels of alveolar partial pressure under discussion here. To determine whether or not a large A-aDO_2 exists in conscious subjects at 2 atmospheres pressure, samples of arterial blood were obtained in 4 of the 7 subjects who breathed oxygen from the 'new' administration system. In Table 2 (also from McDowall, Ledingham, Jacobson & Norman 1965, in preparation) the values for the last alveolar sample in each subject are given together with the arterial oxygen tension measurements. The arterial samples were obtained at least five minutes after the last alveolar so that these results cannot be used to compute the exact alveolar-arterial gradient present; the figures do, however, demonstrate that large, clinically important, gradients do not exist at 2 atmospheres of oxygen in subjects with normal lungs after approximately one hour of exposure to this oxygen pressure.

In the anæsthetized patient or animal the situation is very different and alveolar-arterial gradients for oxygen of between 300 and 400 mmHg routinely occur at 2 atmospheres of oxygen. In Table 3, the percentages of the cardiac output which must have been shunted through unventilated alveoli to produce the alveolar-arterial gradients previously reported by McDowall (1964*a*) in anæsthetized patients and animals at 2 atmospheres have been calculated, assuming an arteriovenous oxygen content difference of 3·5 vols % (Nunn 1964). The calculation is based, in this case, on the tenuous assumption that cardiac output is the same at 1 and 2 atmospheres of oxygen in anæsthetized man and dog. None the less it is unlikely that all of the increase in A-aDO_2 gradient on pressurization was due to reduced cardiac output since this would require that the cardiac output fell by over 50% on pressurization.

Table 2

Values for alveolar and arterial pO_2 in 4 of the subjects who breathed oxygen at 2 atmospheres absolute from the 'test' system

	Alveolar pO_2 (mmHg)	*Arterial pO_2 (mmHg)*
Subject 1	1,315	1,275
Subject 6	1,240	1,240
Subject 7	1,230	1,260
Subject 8	1,268	1,240

Table 3

Alveolar-arterial oxygen gradient and the percentage of venous admixture (shunt) in 8 dogs anæsthetized with trichloroethylene and in 3 patients (2 anæsthetized with halothane, 1 with chloroform)

	Normal pressure			*2 atmospheres*	
	A – aDO_2	*Shunt*		*A – aDO_2*	*Shunt*
8 dogs	152 mmHg	13%	8 dogs	322 mmHg	28%
1 patient	155 mmHg	13%	3 patients	372 mmHg	32%

The conclusion then is that at 2 atmospheres of oxygen the amount of venous admixture is increased over that existing at atmospheric pressure in both man and dog, though the extent of the increase has not been established. Such an increase in venous admixture could be explained if anæsthesia potentiated the pulmonary toxicity of oxygen. E G Karasewitch (personal communication) studied the effects of 2 atmospheres of oxygen in conscious dogs; he found that respiratory distress appeared in six to ten hours and that death resulted in ten to fourteen hours. After the appearance of respiratory distress, the arterial oxygen tension (Bahnson & Mathews 1953) and saturation (Paine *et al.* 1941, Smith *et al.* 1963) fall precipitously. At post-mortem the lungs are found to be red and heavy and microscopy shows extensive alveolar consolidation (Binger *et al.* 1927, Cross 1954).

To determine the influence of general anæsthesia on this process, 10 dogs were exposed to 2 atmospheres of oxygen for eleven hours during anæsthesia with halothane; 3 of these animals will not be considered further since they were allowed to breathe spontaneously and developed central respiratory failure which may have been due to hyperbaric oxygen exposure or to prolonged anæsthesia. The other 7 animals were ventilated by intermittent positive pressure respiration throughout the exposure. After eleven hours the chamber was decompressed and the animals sacrificed.

All the animals survived to the end of the experiment except one which died during decompression; in this animal massive mediastinal emphysema was found at post-mortem, presumably due to rupture of the tracheal mucosa by the cuff on the endotracheal tube during decompression. Macroscopic examination of the lungs in all animals showed no abnormality other than patchy atelectasis on the surface of the dependent lung. Microscopy revealed only dilatation of the perivascular lymphatics in the lungs of some of the animals.

The histological picture was strikingly different from the alveolar congestion and consolidation seen in the lungs of conscious animals (Karasewitch, personal communication). This absence of histological evidence of pulmonary toxicity in the anæsthetized dogs was supported by unaltered values for arterial oxygen content throughout the period of exposure. In one animal the arterial oxygen saturation was measured during ventilation with air both before and after the hyperbaric oxygen exposure; there was no fall in saturation following the eleven-hour exposure.

From these studies it seems clear that the increase in A-aDO_2 reported in anæsthetized patients and animals on pressurization to 2 atmospheres absolute cannot be due to an increased susceptibility to oxygen poisoning produced by general anæsthesia. Further work is required to elucidate the mechanisms involved.

Cerebral Available Oxygen

The amount of oxygen made available to the brain in unit time is the product of the cerebral blood flow and the arterial oxygen content. Arterial oxygen content is increased by exposure to hyperbaric oxygen, although the increase is relatively small unless the arterial blood is desaturated before exposure. It is well established that cerebral blood flow is reduced by oxygen both at normal pressure (Kety & Schmidt 1948) and at raised pressure (Jacobson *et al.* 1963, Lambertsen *et al.* 1953). As a result of these opposing influences exposure to hyperbaric oxygen produces only a small increase in cerebral available oxygen in the healthy human subject or experimental animal (Jacobson *et al.* 1963, Lambertsen *et al.* 1953, McDowall 1964*b*).

The concept that oxygen, especially under pressure, may constrict cerebral vessels led to misgivings about the therapeutic use of hyperbaric oxygen in patients whose cerebral blood flow had been impaired by disease. In an attempt to study this problem, Harper *et al.* (1965) studied the effects of 2 atmospheres of oxygen on the blood flow through the cerebral cortex of dogs after cerebral blood flow had been lowered by reducing the mean arterial blood pressure to 50 mmHg (by controlled bleeding). It was found that under these circumstances hyperbaric oxygen did not produce a further fall in cerebral cortical blood flow. From this evidence it seems that the existence of cerebral tissue hypoxia overrides the constrictive influence of oxygen on the cerebral blood vessels.

These workers also measured the changes which occurred in the oxygen uptake of the cerebral cortex in these animals. Reducing the arterial blood pressure caused a 27% fall in oxygen uptake but the administration of 2 atmospheres of oxygen returned the oxygen uptake of the cerebral cortex to normal despite continuing hypotensive impairment of blood flow.

The influence of anæsthetic drugs on cerebral blood flow must be remembered when considering cerebral available oxygen. McDowall, Harper & Jacobson (unpublished observations) found that the administration of chloroform increased blood flow through the cerebral cortex of the dog by 19% and increased the saturation of cerebral venous blood from a mean value of 60% to one of 71%. This vasodilator action of chloroform has been utilized to raise the cerebral oxygen tension in 2 patients undergoing carotid endarterectomy at 2 atmospheres of oxygen (McDowall *et al.* 1965). Table 4 shows that during anæsthesia with chloroform in oxygen at normal pressure the oxygen tension in the jugular venous blood was 65 and 72 mmHg in these patients. These results may be compared with the value of 40 mmHg reported by Lambertsen *et al.* (1953) for conscious humans breathing 100% oxygen and of 53·7 mmHg reported by Wollman *et al.* (1964) in humans anæsthetized with halothane in oxygen. These results therefore support the findings of the animal study that chloroform increases cerebral blood flow. On pressurizing the chamber to

Table 4

Results in 2 patients anæsthetized with chloroform at 1 and 2 atmospheres of oxygen

Patient	*Pressure (mmHg)*	*Arterial pO_2 (mmHg)*	*Jugular venous pO_2 (mmHg)*	*Arterial pCO_2 (mmHg)*	*Systolic blood pressure (mmHg)*
J F	760	–	72	33	100
	1,520	1,106	125	25	130
	1,520 after endarterectomy	1,165	240	29	–
S I	760	500	65	43	170
	1,520	–	150	42	180

2 atmospheres absolute the oxygen tension of the jugular venous blood in these patients rose to 150 and 125 mmHg respectively, indicating almost complete saturation of cerebral venous blood.

The point of this study is not, necessarily, to advocate the use of chloroform anæsthesia but to emphasize the importance of the choice of anæsthetic technique in the application of hyperbaric oxygen to surgery.

Summary

An efficient system of oxygen administration is fundamental to the clinical application of hyperbaric oxygen therapy. When high alveolar partial pressures of oxygen are obtained in conscious subjects, the alveolar-arterial oxygen gradient is relatively small, at least at 2 atmospheres of oxygen during one hour of exposure.

On the other hand, large alveolar-arterial oxygen gradients exist during anæsthesia at increased pressure but it appears, from studies in anæsthetized dogs, that these large gradients are not evidence that susceptibility to pulmonary oxygen toxicity is increased by general anæsthesia.

Cerebral blood flow is reduced by oxygen though this does not appear to occur when the brain is imperilled by tissue hypoxia. The use of cerebral vasodilators, e.g. chloroform, may be indicated in conjunction with hyperbaric oxygen in certain clinical situations.

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REFERENCES

Bahnson H T & Mathews C M (1953) *Amer. J. Physiol.* **175**, 87

Binger C A L, Faulkner J M & Moore R L (1927) *J. exp. Med.* **45**, 849

Cross F S (1954) *Surgery* **36**, 1001

Harper A M, McDowall D G & Ledingham I McA (1965) Proceedings of the Second International Congress of the Clinical Application of Hyperbaric Oxygen. Ed. C F W Illingworth & I McA Ledingham. Edinburgh (in press)

Jacobson I, Harper A M & McDowall D G (1963) *Lancet* ii, 549

Kety S S & Schmidt C F (1948) *J. clin. Invest.* **27**, 484

Kory R C, Bergmann J C, Sweet R D & Smith J R (1962) *J. Amer. med. Ass.* **179**, 767

Lambertsen C J, Kough R H, Cooper D Y, Emmel G L, Loeschcke H H & Schmidt C F (1953) *J. appl. Physiol.* **5**, 471

McDowall D G
(1964*a*) *Anæsthesia* **19**, 321
(1964*b*) *Brit. J. Anæsth.* **36**, 563

McDowall D G, Jennett W B, Bloor K & Ledingham I McA (1965) The effect of hyperbaric oxygen on the oxygen tension of the brain during chloroform anæsthesia (in preparation)

Nunn J F (1964) *Brit. J. Anæsth.* **36**, 327

Paine J R, Keys A & Lynn D (1941) *Amer. J. Physiol.* **133**, P406

Smith C W, Lehan P H & Monks J J (1963) *J. appl. Physiol.* **18**, 849

Wollman H, Alexander S C, Cohen P J, Chase P E, Melman E & Behar M G (1964) *Anesthesiology* **25**, 180